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TRANSESOPHAGEAL ECHOCARDIOGRAPHIC EVALUATION OF CORONARY ARTERY DISEASE

Dissertation submitted for DM
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AND
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CERTIFICATE

This is to certify that this dissertation titled "**TRANSESOPHAGEAL ECHOCARDIOGRAPHIC EVALUATION OF CORONARY ARTERY DISEASE"** submitted by **Dr.K.SABAPATHY**, to the faculty of Cardiology, The Tamil Nadu Dr.M.G.R.Medical University, Chennai in partial fulfillment of the requirement for the award of DM degree Branch [Cardiology] is a bonafide research work carried out by him under our direct supervision and guidance.

DEAN

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DECLARATION

I, **Dr.K.SABAPATHY** solemnly declare that I carried out this work on "**TRANSESOPHAGEAL ECHOCARDIOGRAPHIC EVALUATION OF CORONARY ARTERY DISEASE"** at Department of Cardiology, Government Rajaji Hospital during the period of January 2006 – January 2008.

I also declare this bonafide work or a part of this work was not submitted by me or any other for any award, degree, diploma to any university, board either in India or abroad.

This is submitted to **The TamilNadu Dr.M.G.R. Medical University, Chennai** in partial fulfillment of the rules and regulation for the DM Cardiology Degree examination.

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Dr. K.SABAPATHY

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INTRODUCTION

With the development of digital imaging capabilities, the emergence of multiplane TEE, and cine loop review, TEE has improved visualization of the coronary artery tree and enhanced detection of its abnormalities. Although TEE is unlikely to replace coronary angiography as the primary imaging modality in coronary artery disease, published reports over the past 12 years have defined a role for TEE in coronary artery disease.

Although the diagnosis of ostial stenosis of the left main coronary artery usually is made by coronary angiography, positioning of the catheter across the obstruction may obscure this diagnosis during the contrast injection. Several authors have reported cases in which TEE was able to make a correct diagnosis in patients with possible left main coronary artery stenosis, in whom damping of the left main coronary artery wave form during catheterization did not allow differentiation of significant left main artery.

The diagnosis of left main coronary artery disease is important in the management of patients with symptomatic coronary artery disease. TEE can be considered when cardiac catheterization suggests ostial stenosis but angiography is inconclusive. The diagnosis of a proximal stenosis may be confirmed by direct visualization of the area of narrowing and supported by color flow Doppler demonstration of flow aliasing.

Recent advances in TEE have enabled the direct evaluation of coronary flow especially in the proximal and mid LAD where the culprit lesion of AWMI is present. TEE can potentially enable direct evaluation of coronary perfusion at a site just distal to culprit lesion in AWMI. We hypothesized that good reperfusion would be associated with less reduction in coronary

flow and therefore would have better ante grade flow visualization by color TEE and less reduced ante grade flow velocity by pulsed TEE.

Atherosclerotic plaque tends to occur focally typically in a certain predisposed regions while sparing adjacent segments. They are scatter along the entire aorta and its main branches e.g. coronary, renal and cerebral vessels.

The close proximity of the TEE probe to the thoracic aorta allows high frequency imaging of the intraluminal structures including detection of atherosclerotic plaque. Atherosclerotic aortic plaque detected by TEE could be used as a marker for coronary artery disease.

In this study we are using TEE to evaluate coronary artery disease by visualizing the proximal left coronary artery, the flow velocity in LAD, and the presence of atherosclerotic plaque in descending thoracic aorta.

AIM

1. Visualization and measurement of the size of Left main coronary in patients with coronary artery disease by Transesophageal echocardiography.
2. Measurement of blood flow velocity in Left main and Proximal Left Anterior Descending artery in patients with coronary artery disease by Transesophageal echocardiography.
3. Measurement of intimal thickness and presence of plaque and its size in descending thoracic aorta in patients with coronary artery disease by Transesophageal echocardiography.

REVIEW OF LITERATURE

Weyman et al first reported the use of two-dimensional transthoracic echocardiography to visualize the coronary arteries more than 20 years ago. In adults, surface imaging provides morphologic information usually limited to the proximal left coronary artery and the origin of the right coronary artery.

Zwicky et al first reported the use of trans-esophageal echocardiography (TEE) to evaluate the coronary arteries. The advantages of the transesophageal approach over the transthoracic approach in coronary artery imaging include the absence of anatomic obstacles between the ultrasound transducer and the aortic root and better resolution characteristics using higher-frequency (7-MHz) transducers.

The use of color flow Doppler mapping and spectral Doppler echocardiography to measure coronary artery flow velocities during TEE was first reported by **Yamagishi et al.**

With TEE, the coronary arteries are visualized primarily in the short-axis plane at a transducer angle of 0°. The normally placed ostium of the left main coronary artery is visualized with the transducer positioned just above the aortic cusps and rotated slightly to the left. The left main coronary artery, arising from the corresponding sinus, is usually 0.5 to 2.5 cm in length. Small adjustments in the transducer orientation are often necessary to view the vessel along its full length from the aortic root to its bifurcation. The bifurcation is usually Y-shaped with the left anterior descending (LAD) artery coursing to wards the apex and the left circumflex in the atrioventricular groove. Further adjustments in transducer orientation may be required to optimize visualization of the proximal left anterior descending coronary artery. The color flow Doppler signal in the LAD can be used to guide placement of the pulsed Doppler

sample volume for flow velocity interrogation.

Normally, the ostium of the right coronary artery arises at the 7 0'clock position on the short-axis view of the aorta and is visualized as the probe is withdrawn 1 to 2 cm above the level of the aortic valve and is flexed anteriorly. Further anterior flexion permits visualization of the right coronary artery as it courses downward and to the right. The right coronary ostia and 1 to 2 cm segment of the proximal right coronary artery also can be visualized in the longitudinal plane typically at 110° to 130° of rotation. Minor angle adjustments of the multiplane may facilitate imaging of the coronary arteries. Color flow Doppler flow interrogation of the right coronary artery is possible; however, spectral Doppler is usually not possible because of suboptimal beam orientation.

Two-dimensional imaging of the left main coronary artery is enhanced by the near perpendicular alignment of the ultrasound beam with the left main coronary artery, relying primarily on axial resolution. Proper gain adjustment is crucial to imaging and operators should be careful not to under gain the images. The color-coded Doppler flow pattern may help identify pathology of the coronary arteries. Appropriate Nyquist limits for color flow Doppler mapping in the coronary arteries are in the range of 0.5 meter per second. If the Nyquist limits are set inappropriately low, areas of flow aliasing may be evident in the absence of stenosis, potentially increasing the number of false positives. With these caveats, color flow channel delineation of narrowing increases the sensitivity and specificity of the diagnosis of a stenosis.

In the study **Yoshida et al** using biplane TEE, sensitivity was 91% and specificity 100% for the detection of proximal coronary artery luminal narrowing. Negative predictive accuracy was 98% and positive predictive accuracy 100%. In the studies by **Tradif et al** and **Khandheria et al** using multiplane TEE, the sensitivity and specificity for detection of

proximal coronary narrowing were 100% when the results were compared with the angiographic data.

Pulsed Doppler and color Doppler echocardiography combined with TEE can be used to measure the velocity of the proximal coronary artery blood flow. The left main and left circumflex coronary arteries are nearly perpendicular to the echo beam, whereas the left anterior descending coronary arteries are nearly parallel to it. The velocity measurement is dependent on the position of the sample volume in relation to the coronary luminal narrowing. The velocity is normal proximal to the stenosis, increased within the stenosis, and reduced distally. TEE also can noninvasively evaluate the functional results of the left anterior descending coronary artery after an angioplasty. TEE can evaluate the stenosis of the just proximal portion of the right coronary artery; however, because the right coronary artery is not parallel to the echo beam and highly movable, it is very difficult to measure the flow velocities.

Yamagishi et al reported that coronary blood flow could be detected and measured during TEE in only 77% of patients. The development of multiplane TEE and intravenous contrast agents has increased the efficacy of TEE. Coronary flow velocity is best measured with pulsed Doppler interrogation of the very proximal segment of the left anterior descending coronary artery. Flow measurements in the right coronary artery are limited. Because of transitional motion, the left anterior descending coronary artery does not lie in the same position throughout the entire cardiac cycle. The position of the left anterior descending coronary artery is more stable in diastole.

Doppler sample volume using the position of the vessel is in diastole. The probe angle and sample volume are adjusted in order to orient the Doppler signal parallel to coronary flow. Color flow can aid in positioning of the pulsed Doppler sample volume.

The characteristic normal flow pattern within the left anterior descending coronary artery consists of a minor systolic flow signal and a major diastolic signal due to extra vascular compression during systole component. This flow pattern is very similar to that of the Doppler-tip catheter or Doppler-tip guide wire. The basal coronary flow pattern is influenced by the presence of cardiac disease. Patients with severe aortic stenosis have a depressed systolic coronary flow velocity. This abnormal systolic component of coronary flow velocity in severe aortic stenosis may result from reduced aortic pressure during systole. For example, patients with long-standing hypertension, with or without left ventricular hypertrophy, have higher baseline coronary flow velocities. These high velocities may result in increased intimal shear stress. Because coronary flow is physiologically predominant during diastole, it is unlikely that a reduction in systolic flow alone may result in myocardial ischemia.

Baseline coronary flow velocity is an important determinant of the CFR. The normal range of baseline diastolic velocity of coronary artery flow is less than 60 centimeters per second. Patients with higher basal coronary flow may have diminished CFR. Using TEE, **Memmola et al** demonstrated altered coronary flow dynamics in patients with obstructive hypertrophic cardiomyopathy. Reduced or reversed systolic and increased diastolic coronary flow velocity with consequently higher diastolic and systolic coronary flow velocity ratio were associated with impaired coronary flow reserve**. Kisanuki et al** showed reduced CFR during systole and diastole in patients with moderate-to-severe chronic aortic regurgitation compared with the control group. Similarly, in these patients, baseline coronary flow velocity was increased.

With technical advancements, including high frequency, multiplane transducers, digital acquisition and display, and left-sided contrast agents, TEE is emerging as a promising method

for evaluating coronary artery disease. Visualization of proximal coronary artery stenoses and coronary artery anomalies is already possible. Research studies using TEE measurement have contributed to under standing coronary artery physiology and may prove to be a valuable clinical tool in the future.

TEE would also enable the non invasive differentiation of TIMI-3 flow from TIMI 2 in patients with AWMI in acute phase. Mere presence of antegrade flow in proximal left coronary generally indicates TIMI 2 or 3 flow. In patients with TIMI 3 flow the LAD flow velocity is present with even it may be increased.

Intra coronary Doppler guidewire studies showed that coronary blood flow velocity is preserved or even increased in patients with TIMI 3 flow with a diastolic peak velocity of 36 ± 11 cm/sec whereas flow velocity is reduced in patients with TIMI 0 to 2 flow. Therefore for most patients TIMI 3 flow can be diagnosed on the basis of higher peak diastolic LAD flow velocity TIMI 2 or less by low flow velocity.

Coronary artery atherosclerosis is the principal cause of coronary artery disease and is the single largest killer of both men and women in the world . A major recent advance has been a refined understanding of the nature of atherosclerotic plaque and the phenomenon of plaque rupture, which is the prime cause of acute coronary syndrome (ACS) and AMI. In many cases (perhaps more than half), the plaque that ruptures and results in the clinical syndromes of ACS and AMI is less than 50% occlusive. These so-called vulnerable plaques, as compared with stable plaques, consist of a large lipid core and thin, fibrous caps and are subjected to greater biomechanical stress, thus leading to rupture that perpetuates thrombosis and ACS.

Coronary artery atherosclerosis or CAD refers to the presence of atherosclerotic changes within the walls of the coronary arteries, which causes impairment or obstruction of normal

blood flow with resultant myocardial ischemia. CAD is a progressive disease process that generally begins in childhood and manifests clinically in mid-to-late adulthood. The distribution of lipid and connective tissue in the atherosclerotic lesions determines whether they are stable or at risk of rupture, thrombosis, and clinical sequelae.

A healthy endothelial layer is thrombo resistant because of the production of heparin sulfate and eicosanoids, which inhibit thrombin activation and platelet adhesion, respectively. Endothelial cells also produce relaxation factors (eg, endothelium-derived relaxing factor [EDRF] or nitric oxide) and vasoconstricting factors (endothelin) that affect the resting tone of the underlying media containing several layers of smooth muscle cells (SMCs). The media are bound on the outside by an external elastic lamina that separates them from the adventitia, which consists mainly of fibroblasts, SMCs, and a matrix containing collagen and proteoglycans.

Over the past decade, **Fuster et al** and colleagues have proposed that vascular injury starts the atherosclerotic process. According to their response-to-vascular injury theory, injury to the endothelium by local disturbances of blood flow at angulated or branch points, along with systemic risk factors (eg, hyperglycemia, dyslipidemia, cigarette smoking, possibly infection) perpetuates a series of events that culminate in the development of atherosclerotic plaque.

Atherosclerotic plaque may require 10-15 years for full development. Further growth is determined by the local activity of regulatory substances (ie, interleukin (IL)–1, IL-6, transforming growth factor-beta) and by thrombin, leukotriene, prostaglandin, fibrin, and fibrinogen.

Although a logical conclusion is that the most severely stenotic lesions are the ones at

the greatest risk of sudden occlusion, this is not the case. As previously described, ACS has been shown to more often develop because of rupture and thrombosis of mild (<60%) coronary stenoses. This occurs because of the relatively higher lipid content of the lipid core, the thinner fibrous cap, and the increased leukocyte activity at the shoulder regions of the plaque. These characteristics make such plaques, called the vulnerable plaques, much more prone to rupture.

Mechanisms of the effects of risk factors

The presence of risk factors accelerates the rate of development of atherosclerosis. Smoking increases platelet activity and catecholamine levels, alters prostaglandins, and decreases high-density lipoprotein (HDL) levels. Hypertension causes endothelial dysfunction and increases collagen, elastin, and endothelial permeability and platelet and monocyte accumulation. Diabetes causes endothelial dysfunction, decreases endothelial thrombo-resistance, and increases platelet activity, thus accelerating atherosclerosis.

Plaque growth and vascular remodeling

As endothelial injury and inflammation progress, fibroatheromas grow and form the plaque. As the plaque grows, 2 types of remodeling occur, (1) positive remodeling and (2) negative remodeling.

Positive remodeling

Positive remodeling is an outward compensatory remodeling (the **Glagov phenomenon**) in which the arterial wall bulges outward and the lumen remains uncompromised. Such plaques grow further, although they usually do not cause angina because they do not become hemodynamically significant for a long time. In fact, the plaque does not begin to encroach on the lumen until it occupies 40% of the cross-sectional area. The encroachment must be 70% or greater to cause flow limitation. Such positively remodeled lesions thus form the bulk of the

vulnerable plaques, grow for years, and are more prone to result in plaque rupture and ACS than stable angina, as documented by intravascular ultrasound (IVUS) studies.

Negative remodeling

Many fewer lesions exhibit almost no compensatory vascular dilation, and the atheroma steadily grows inward, causing gradual luminal narrowing. Many of the plaques with initial positive remodeling eventually progress to the negative remodeling stage, causing narrowing of the vascular lumen. Such plaques usually lead to the development of stable angina. They are also vulnerable to plaque rupture and thrombosis.

Eruption of the vulnerable plaque

Tight coronary atheromas rarely cause ACS and MI. In fact, most of the atheromas that cause ACS are less than 50% occlusive as demonstrated by coronary arteriography. Atheromas (plaques) with smaller obstruction experience greater wall tension, which changes in direct proportion to their radii. Most plaque ruptures occur because of disruption of the fibrous cap, which allows contact between the highly thrombogenic lipid core and the blood. These modestly obstructive plaques, which have a greater burden of soft lipid core and thinner fibrous caps with chemoactive cellular infiltration near the shoulder region, are called vulnerable plaques. The amount of collagen in the fibrous cap depends on the balance between synthesis and destruction of intercellular matrix and inflammatory cell activation.

Imaging Studies

Transthoracic echocardiography helps to assess left ventricular function, wall motion abnormalities in the setting of ACS or AMI, and mechanical complications of AMI. Transesophageal echocardiography is most often used for assessing possible aortic dissection in the setting of AMI. Stress echocardiography can be used to evaluate hemodynamically

significant stenoses in stable patients who are thought to have CAD. Treadmill echocardiography stress testing and dobutamine echocardiography stress testing provide equivalent predictive values.

Nuclear imaging studies (myocardial perfusion imaging) are also useful in assessing patients for hemodynamically significant coronary artery stenoses. Stress and rest nuclear scintigraphic studies using thallium, sestamibi, or teboroxime are sometimes helpful. Radionuclide stress myocardial perfusion imaging can be used to quantify coronary flow reserve (CFR). Likewise Magnetic resonance angiography and Electron beam CT scanning plays significant role in imaging coronaries in this setup.

Coronary angiography remains the criterion standard for defining significant flow-limiting stenoses that must be revascularized through percutaneous or surgical intervention to improve prognosis. Quantitative coronary angiography (QCA) is used to perform computerized quantitative analysis of the entire coronary tree. It introduces a correction factor for the presence of diffuse disease. QCA has been widely used in many trials of atherosclerotic progression and regression.

Limitations of coronary arteriography are as follows:

Severity of stenosis is generally estimated visually, but estimation is limited by the fact that interobserver variability may range from 30-60%. The presence of diffuse disease also may lead to underestimation of stenoses because the stenosed areas are expressed as a percent of luminal diameter compared with adjacent normal coronary segments and in diffuse disease, no such segments exist. This usually occurs in diabetic patients, in whom coronary arteries are traditionally described as small-caliber vessels, when that appearance is actually due to the presence of diffuse symmetrical involvement of the entire vessel, as elucidated by recent IVUS

studies.

Because of the inherent limitations of coronary angiography, attention has been directed to using physiological approaches for determining the severity of coronary stenoses.

The 5 methods of measuring human coronary blood flow in the cardiac catheterization laboratory are (1) thermodilution, (2) digital subtraction angiography, (3) electromagnetic flow meters, (4) Doppler velocity probes (for measuring CFR), and (5) pressure wires (for measuring fractional flow reserve [FFR]). Although most current methods measure relative changes in coronary blood flow, useful information about the physiological significance of stenosis, cardiac hypertrophy, and pharmacological interventions can be obtained from these measurements.

Doppler velocity probes use a Doppler flow meter, which is based on the principle of the Doppler effect. This is the most widely applied technique for measuring coronary flow in humans. High-frequency sound waves are reflected from moving red blood cells and undergo a shift in sound frequency proportional to the velocity of the blood flow.

In pulsed-wave Doppler methods, a single piezoelectric crystal can both transmit and receive high-frequency sound waves. These methods have been successfully applied in humans by using miniaturized crystals fixed to the tip of catheters. Technological developments have further miniaturized steerable 12-MHz Doppler guidewires to a diameter of 0.014 inches.

Flow due to a stenosis can therefore be assessed distally and proximally. The Doppler guidewire measures phasic flow velocity patterns and tracks linearly with flow rates in small, straight coronary arteries.

Indications for Doppler velocity probe use include determining the severity of intermediate stenosis (40-60%) and for evaluating whether normal blood flow has been

restored after PTCA. The use of smaller Doppler catheters allows measurement of selective coronary artery flow velocity. By noting the increase in flow velocity following administration of a strong coronary vasodilator, such as papaverine or adenosine, the CFR can be defined. CFR provides an index of the functional significance of coronary lesions that obviates some of the ambiguity of anatomical description.

The current Doppler probe method has limitations. Limitations include (1) only changes in flow velocity, rather than absolute velocity or volumetric flow, are measurable; (2) the change in flow velocity is directly proportional to changes in volumetric flow only when vessel dimensions are constant at the site of the sample volume; (3) other factors, including left ventricular hypertrophy and myocardial scarring, can also affect CFR; and (4) changes in luminal diameter and arterial cross-sectional area during interventions are not reflected in measurements of flow velocity, thus potentially causing underestimation of the true volume flow.

Basic Concepts

Spectrum Analysis

If blood flow were continuous rather than pulsatile, if blood vessels followed straight lines and were uniform in caliber, if blood flowed at the same velocity at the periphery as well as in the center of the lumen, and if vessels were disease free, then each blood vessel would produce a single Doppler ultrasound frequency. However, blood flow is pulsatile, vessels are not always straight or uniform in size, flow is slower at the periphery than in the center of the vessel, and the vessel lumen may be distorted by atherosclerosis and other pathology.

For these reasons, blood flow produces a mixture of Doppler frequency shifts that changes from moment to moment and from place to place within the vessel lumen.

Spectrum analysis is needed to sort out the jumble of Doppler frequencies generated by blood flow and to provide quantitative information that is critical for diagnosis of vascular pathology.

The Doppler Spectrum

The word spectrum, as derived from Latin, means image. Doppler spectrum as an image is a graph showing the mixture of Doppler frequencies present in a small area of a vessel over a short period of time. The key elements of the Doppler spectrum are time, frequency, velocity and Doppler signal power.

The Power Spectrum

Doppler frequency spectrum is some times called a power spectrum, because the power, or strength of each frequency is shown by the brightness of the pixels. The power of a given frequency shift, in turn, is proportionate to the number of red blood cells producing that frequency shift. If a large number of blood cells are moving at a certain velocity, the corresponding Doppler frequency shift is powerful, and the pixels assigned to that frequency are bright. Conversely, if only a small number of cells are causing a certain frequency shift, the pixels assigned to that frequency are dim.

Frequency versus Velocity

The echoes that return to the transducer from a blood vessel contain only Doppler frequency shift information; yet the Doppler spectrum often displays both velocity (cm/sec or m/sec) and frequency (kHz) information.

If the instrument “knows” the Doppler angle, it can then compute the blood flow velocity via the Doppler formula. When the operator informs the ultrasound machine of this angle, the frequency shift is proportional to blood flow velocity. The frequency spectrum

becomes a velocity spectrum. It is generally recommended that the Doppler angle should be between 45 and 60 degrees for greatest accuracy.

The Doppler frequency shift observed in a tortuous artery might be radically different from one point to another, but angle-corrected velocity measurements will be similar throughout the vessel, in spite of dramatic changes in vessel orientation relative to the skin. To determine stenosis severity, different diagnostic parameters would be needed for different ultrasound transducers (e.g., 3.5, 5, or 7.5 MHz).

The sample volume

The frequency spectrum shows blood flow information from a specific location called the Doppler sample volume and the following are the three characteristics of the Doppler sample volume. First, it is in fact, a volume (three dimensions), even though only two of its dimensions are shown on the duplex image. The “thickness” of the sample volume cannot be shown on the two-dimensional spectrum display, and this can sometimes lead to errors of localization. Second, the actual shape and size of the sample volume may be somewhat different from the linear representation shown on the duplex image. Third, and most important, the Doppler spectrum display flow information only within the sample volume and does not provide information about flow in other portions of the blood vessel that are visible on the ultrasound image.

Flow Direction

The frequency spectrum shows blood flow relative to the transducer. Flow in one direction, with respect to the transducer, is displayed above the spectrum baseline, and flow in the opposite direction is shown below the baseline. The flow direction is relative to the transducer and is not absolute. When accurate determination of flow direction is necessary, a

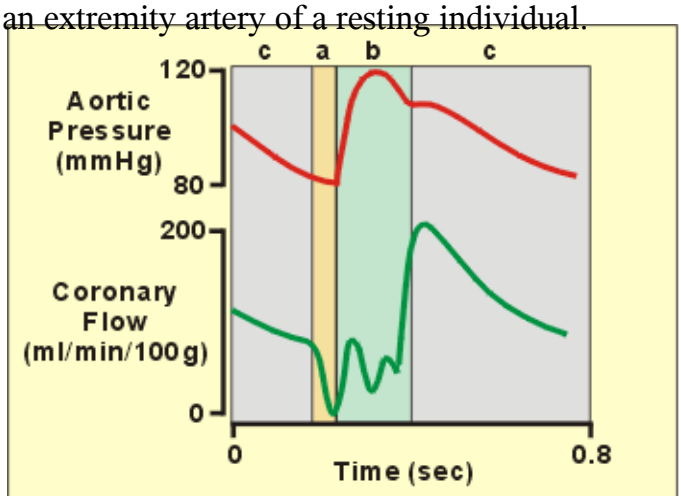
comparison should be made with a reference vessel in which the flow direction is known.

In arteries, each cycle of cardiac activity produces a distinct “wave” on the Doppler frequency spectrum that begins with systole and terminates at the end of diastole. The term waveform refers to the shape of each of these waves, and this shape, in turn, defines a very important flow property called pulsatility.

Low-pulsatility Doppler waveforms have broad systolic peaks and forward flow throughout diastole. The carotid, vertebral, renal, and celiac arteries all have low-pulsatility waveforms in normal individuals because these vessels feed circulatory systems with low resistance to flow.

Moderate pulsatility Doppler waveforms have an appearance somewhere between the low and high resistance pattern. With moderate flow resistance, the systolic peak is tall and sharp, but forward flow is present throughout diastole (perhaps interrupted by early-diastolic flow reversal). Examples of moderate pulsatility are found in the external carotid artery and the superior mesenteric artery .

High – pulsatility Doppler waveforms have tall, narrow, sharp systolic peaks and reversed or absent diastolic flow. The classic example of high pulsatility is the triphasic flow pattern seen in an extremity artery of a resting individual.



Pulsatile nature of left coronary artery blood flow. Flow is lower during phases of isovolumetric contraction (a) and ejection (b) than during diastole (c).

Pulsatility and flow resistance may be gauged qualitatively, either by visual inspection of the Doppler spectrum waveforms or by listening to the auditory output of a Doppler instrument. Qualitative assessment of pulsatility is often sufficient for clinical vascular diagnosis, but in some situations (e.g., assessment of renal transplant rejection), quantitative assessment is desirable. A variety of mathematical formulae can be used for this purpose, but the most popular measurements are the pulsatility index (of gosling), the resistivity index (of Pourcelot), and the systolic/diastolic ratio.

Both physiology and pathology may alter arterial pulsatility proper interpretation of pulsatility requires knowledge of the normal waveform characteristics of a given vessel and the physiologic status of the circulation at the time of examination. The status of cardiac function is also important; showed ventricular emptying, valvular reflux, valvular stenosis, and other factors may significantly affect arterial pulsatility.

Severe arterial obstruction is present proximal (upstream) to the point of Doppler examination, systolic flow acceleration may be slowed substantially.

Laminar and Disturbed Flow

Blood generally flows through arteries in an orderly way, with blood in the center of the vessel moving faster than the blood at the periphery. This flow pattern is described as laminar, because the movement of blood is in parallel lines. When flow is laminar, the great majority of blood cells are moving at a uniform speed, and the Doppler spectrum shows a thin line that outlines a clear space called the spectral window.

In disturbed flow, the movement of blood cells is less uniform and orderly than in laminar flow. Disturbed flow is manifested as spectral broadening or widening of the spectral

waveform. The degree of spectral broadening is proportionate to the severity of the flow disturbance. Although disturbed blood flow often indicates vascular disease, it must be recognized that flow disturbances also occur in normal vessels. Kinks, curves, and arterial branching may produce normal flow disturbances.

Volume Flow

This is done by measuring the average flow velocity across the entire lumen while simultaneously measuring the vessel diameter, which is converted mathematically into cross-sectional area. Knowing the average velocity and the vessel area, it is an easy matter for the Doppler instrument to calculate the blood flow (in ml /min), and this is done automatically by the ultrasound instrument.

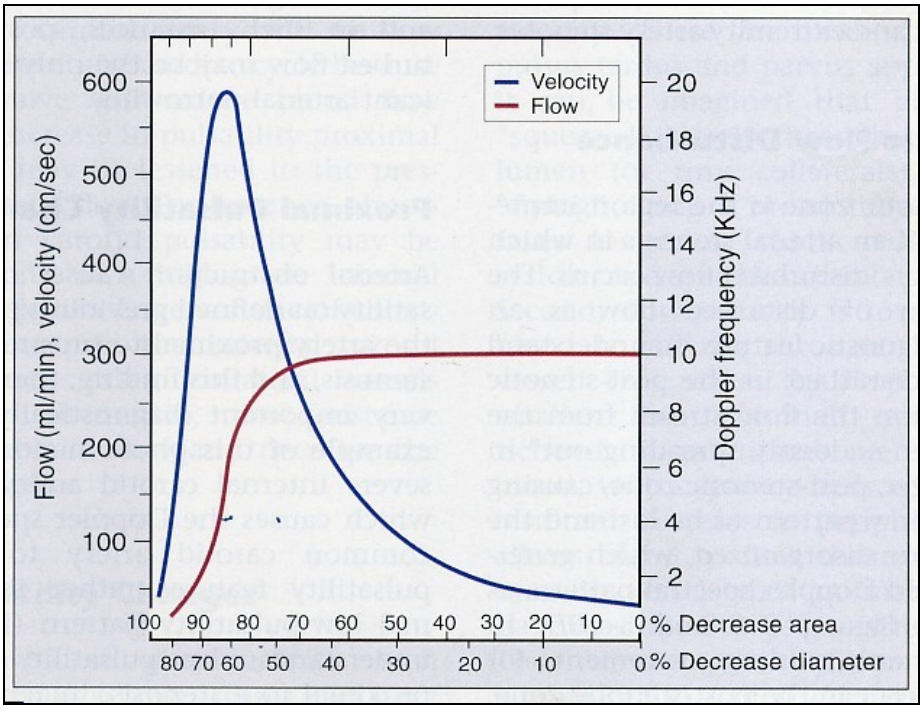
Five main categories of information are used in this process: (1) increased setnotic zone velocity, (2) disturbed flow in the poststenotic zone, (3) Proximal pulsatility changes, (4) distal pulsatility changes, and (5) indirect effects of obstruction, such as collateralization.

Increased Stenotic Zone Velocity

The term stenotic zone refers to the narrowed portion of the arterial lumen. For determining the severity of arterial stenosis, the single most valuable Doppler finding is increased velocity in the stenotic zone. Flow velocity is increased in the stenotic zone because blood must move more quickly if the same volume is to flow through the narrowed lumer as through the larger, normal lumen. The increase in stenotic zone velocity is directly proportional to the severity of luminal narrowing.

Three stenotic zone velocity measurements are commonly used to determine the severity of arterial stenosis: (1) peak systolic velocity (also called peak systole), which is the highest systolic velocity within the stenosis; (2) end-diastolic velocity (also called end diastole), which

is the highest end-diastolic velocity; and (3) the systolic velocity ratio, which compares peak systole in the stenosis with peak systole proximal to the stenosis (in a normal portion of the vessels).



Peak systole in the stenotic zone is the first Doppler parameter to become abnormal as an arterial lumen becomes narrowed. The region of maximum velocity within the stenotic zone may be quite small, and for that reason, the sonographer must “search” the stenotic lumen with the sample volume to locate the highest flow velocity. If the highest flow velocity is overlooked, the degree of stenosis may be underestimated. As shown in peak systole rises steadily with progressive narrowing, but ultimately the flow resistance becomes so high 9at about 80% diameter reduction) that peak systole falls to normal or even subnormal levels. This drop in velocity can cause the unwary to underestimate the severity of a high-grade stenosis. Low flow velocity in a very high-grade stenosis may also lead to false diagnosis of arterial occlusion, if the velocity is so low that Doppler signals cannot be detected with ultrasound.

The end-diastolic velocity (end diastole) in the stenotic zone, generally remains normal with less than 50% (diameter) narrowing, as there is not pressure gradient across the stenosis in diastole. With moderate stenosis (505-70% diameter reduction), however, a pressure gradient exists throughout diastole, and end-diastolic velocity is elevated in proportion to stenosis severity. With severe stenosis (70%-90% diameter reduction), a substantial pressure gradient exists throughout diastole, and diastolic velocities are high. Furthermore, with progression of stenosis severity, end diastolic velocity increases at a greater rate, proportionately, than the peak systolic velocity, and as a result, the difference between peak systolic and end-diastolic velocity decreases. End-diastolic velocity, therefore, is a particularly good marker for severe stenosis.

The systolic velocity ratio, as defined previously, is an additional important parameter for the diagnosis of arterial stenosis. This parameter is used to compensate for patient-to-patient hemodynamic variables, such as cardiac function, heart rate, blood pressure, and arterial compliance, Tachycardia, for instance, tends to increase peak systole in the stenotic zone, whereas poor myocardial function may decrease peak systole. The systolic velocity ratio allows the patient to act as his or her own physiologic “standard,” because peak systole in the stenotic zone is compared with peak systole in a normal arterial segment.

The post-stenotic zone is the region immediately beyond an arterial stenosis in which disorganized or “disturbed” flow occurs. Flow is disturbed in the post-stenotic region because the flow stream from the stenotic lumen suddenly spreading out in the much larger, post-stenotic zone, causing the laminar flow pattern to be lost and the flow to become disorganized, which generates a disturbed Doppler spectral pattern. The maximal flow disturbance occurs within 1cm beyond the stenosis, and in very severe stenosis, soft tissues adjacent to this portion

of the artery may vibrate, causing a “visible bruit” on color Doppler images. Approximately 2cm beyond the stenosis, the flow disturbance becomes less violent and spectral broadening diminishes. Severe flow disturbances are “beacons” indicating the presence of arterial disease.

Arterial obstruction causes increased pulsatility (as defined previously) in portions of the artery proximal to (upstream from) the stenosis, and this finding, therefore, may be very important diagnostically.

In systole, flow will go forward for only a brief moment and will then slow abruptly; therefore, the systolic peak will be sharp and narrow. The increase in pulsatility proximal to a stenosis may be lessened in the presence of collateral flow.

Doppler waveform abnormalities seen distal to a stenosis (downstream) also have considerable value in the diagnosis of arterial stenosis. The flow velocity in a normal, wide open, artery increases abruptly in systole, and the systolic peak is reached quickly. The Doppler waveform distal to a severe arterial obstruction has a “damped” appearance. Which means that the acceleration is slowed there are three causes. First, it can be imagined that blood is being “squeezed” slowly through the obstructed lumen (or tiny collaterals), rather than “flying” along a broad tube. Therefore, it takes longer to reach peak velocity. Second, flow velocity is low, because less blood is moving through the obstructed vessel. This makes the Doppler waveform smaller than normal overall. Finally, ischemic distal tissues are “begging” for blood, with capillary beds wide open. The resultant decrease in peripheral resistance allows blood to flow with low velocity. The net effect of all three factors is the damped (also called dampened) waveform appearance.

DIAGNOSIS OF UNSUCCESSFUL THROMBOLYSIS AFTER ACUTE MYOCARDIAL INFARCTION

Trials of thrombolysis and primary angioplasty haves shown that coronary artery patency and flow characteristics following thrombolytic therapy are independent prognostic predictors of the outcome in acute myocardial infarction(AMI). A number of studies suggest that the patency rates with TIMI 3 flow can not be achieved in more than 54% of patients even with the best thrombolytic regimen. The timely detection of this failed reperfusion is very important for the further rational management of patients with AMI. The need is to diagnose it accurately and cost effectively preferably using non-invasive techniques. This has to be followed by prompt and efficient measures to attempt reperfuse these blocked vessels by pharmacological, catheter based or combined strategies.

Cessation of chest pain has been regarded as a clinically predictive sign of reperfusion though its quantification for clinical trials is difficult. Only complete resolution of chest pain is a good predictor and this sign has been reported only in 29% patients with patent arteries. Analgesia which is an important part of the management of AMI can mask this sign. It is therefore necessary to have more objectively defined markers along with resolution of chest pain.

Reperfusion Arrhythmias although observed frequently after thrombolysis and primary PTCA, none of the observed arrhythmias such as accelerated idioventricular rhythm have been shown to add independently to the predictive value of diagnosing reperfusion.

Non resolution of ST-segment changes after thrombolysis has been shown to be a predictor of worse long-term outcome compared with the cohort with good resolution. This analysis is a better predictor of successful perfusion than failed reperfusion. Studies have shown a variety of ECG indices for reperfusion failed or success. These included 25% reduction in ST-segment elevation identified in the 'worst lead' on the 60-180 min post

thrombolytic ECG, the pre to post thrombolysis ECG of maximal ST segment elevation ratio or the sums of post to pre-thrombolysis ST-segment elevation that is equal or less than 0.5. All these indices have been described as having reasonable sensitivity and specificity, irrespective of the infarct site . Continuous ST segment monitoring has also been shown to have a good predictive value for non reperfusion in GUSTO-I study especially when the initial ST segment elevation is more than 4 mm.

Of the numerous markers Creatinine kinase , isoenzymes troponin-T or I and myoglobin measurements have been used extensively for the early diagnosis of AMI. The rapid peaking of myoglobin seems to be the earliest marker of a successful recanalization, while the rate of troponin-T rise over 3 hours post-thrombolysis has revealed very high(94%) sensitivity as well as specificity (100%) in this situation. There is however limited evidence that any of these markers can predict failure to achieve TIMI-3 flow at 60-90 mins with any degree of similar accuracy. At present these assays are mostly reserved for post hoc confirmation rather than direct decision, so that they may not help in the tirage of patients with failed reperfusion. Early peaking of levels of these markers while suggesting restoration of flow doesnot necessarily mean achievement of reperfusion at tissue level (restoration of micro vascular reperfusion). The failure of the accuracy and interpretation makes their reliable use difficult in the setting of failed reperfusion, especially in the absence of concomitant ST segment resolution.

There is some evidence to suggest that patients who fail to achieve detectable fibrinogenolysis following thrombolysis could benefit from additional thrombolysis. In one small study, benefit was confined to those patients whose fibrinogen remained at greater level following therapy with streptokinase'. Although fibrinogen assay is not routinely tested, this measurement is potentially advantageous for distinguishing patients in whom non reperfusion

is primarily due to non-fibrinogenolysis rather than due to no reflow. In such cases it would suggest that further thrombolysis and/or intensified antiplatelet therapy rather than interventional treatment may be beneficial.

Emerging Diagnostic Strategies

Even though Sesta Mffll has good accuracy in assessing patency after systemic thrombolysis, the need to obtain pre thrombolytic scans preclude this method from wide clinical application. The acute assessment of micro vascular perfusion by myocardial contrast echocardiography may be the most promising strategy. Contrast agents are currently being tested in pre clinical trials and are likely to become available for clinical trial assessment shortly.

There is no proven strategy which is clearly superior and can be recommended as the treatment of choice. In the absence of sound clinical data it however seems logical to recommend a careful, frequent assessment of the patient's clinical status after instituting systemic thrombolysis. This should include frequent 12 lead ECG control. A 90-120 run recording is very important which could form a basis for consideration for further management which could be PCI after administering Gp llb/IIIa blockers, if the option is easily achievable. It is likely that patients with failed thrombolysis comprise a heterogeneous group with different levels of failed lysis, micro vascular no reflow or different degrees of critical narrowing in the target or infarct related vessel. Careful evaluation of these factors in the individual patients will result in a more tailored and tiered approach.

Thrombus burden as a cause of TIMI-2 flow.

A potential problem in the treatment of AMI is a possibility of distal embolization, which may be even more important during catheter-based treatment The consequences of

coronary embolism depend on the size (and number) of emboli. The smaller the embolus, the greater the chance that it will migrate distally to a small artery and the less likelihood of myocardial necrosis. For particle sizes ranging from 15 to 45 µm, baseline blood flow into the embolized area may actually increase secondary to reactive hyperemia in the myocardium surrounding the embolized regions Conversely, the larger the embolic particle, the greater the chance that it will lodge proximally in a large coronary artery and result in ischemic myocardial necrosis. Coronary arterial resistance increases if massive embolization of the coronary arterial bed occurs.

Recently, **Gregorini et al** reported that coronary intervention might also cause neurohormonal reflexes and vasoconstriction, thus provoking a reduction in coronary blood flow velocity in patients with AMI. If coronary resistance could play an important role, it is likely that an increase in coronary resistance might even abate over time and, therefore, might possibly manifest as a transient phenomenon.

A rapid deceleration of diastolic CBFV is another characteristic of capillary damage. The intra myocardial blood volume decreases with damage to the capillaries and/or post capillary venules. If the coronary inflow exceeds the capacitance, an impeding effect occurs on the diastolic coronary inflow by altering peri vascular strains, resulting in a rapid decrease in CBFV. This finding is also supported by recent findings that a shorter diastolic deceleration time is associated with poorer tissue perfusion and worse functional outcomes.

Atherosclerosis of the Aorta:

Risk Factor, Risk Marker, or Innocent Bystander?

Atherosclerosis is a diffuse process, it is not surprising that multiple studies have found an association between aortic atherosclerosis and CAD. Extrapolation of these studies to the

general population is misleading due to study limitations which include the highly selected nature of patient groups having TEE and coronary angiography for specific clinical indications .Several authors have shown a strong association between aortic atherosclerosis and other risk factors for stroke. Age, hypertension, atrial fibrillation, carotid artery disease, and diabetes mellitus are well-known risk factors for cardiac events and stroke. These findings challenge previously drawn conclusions about the cause-and-effect relationship of aortic atherosclerosis and clinical outcome.

In the Stroke Prevention in Atrial Fibrillation (SPAF) III trial, complex aortic atherosclerosis emerged as a significant independent predictor for stroke and CAD without adjusting for age. Age has been shown to be a strong independent predictor of aortic atherosclerosis which suggests that atherosclerosis may be a marker of aging rather than a true risk factor for stroke or CAD. One autopsy study concluded that the presence of moderate or severe aortic atherosclerosis did not predict ischemic stroke subtype. Complex atherosclerosis is a high-risk marker in high-risk patients, but it is also a marker of many other risk factors for stroke or CAD, so a cause-and-effect relationship cannot be established This SPARC study is the largest prospective population based TEE study published to date. However, size limitations imposed by the use of an invasive study in a relatively healthy population sample may have limited the ability of the SPARC study to detect a statistically significant hazard for the less prevalent risk factors.

Aortic atherosclerosis is increasingly detected with more frequent use of TEE. It does not appear to be an independent risk factor or a risk marker for vascular events in the general population.

MATERALS AND METHODS

Study Design

This study was conducted to observe the role of Transesophageal echocardiography in assessing the CAD by measuring the size and blood flow pattern in proximal left coronary and to visualize and detect the presence plaque in descending thoracic aorta.

Total Number of Patients

There were totally hundred and six (106) patients were included in this study.

Place of Study

This study was conducted in Government Rajaji Hospital Madurai, hundred and six (106) patients who were admitted in ICCU and fulfilled the inclusion criteria were selected for this study.

Study Period

This study was conducted from January 2006 to January 2008.

Inclusion Criteria

Patients who presented with acute anterior wall myocardial infarction were taken for this study.

All the patients were thrombolysed with intravenous streptokinase.

Exclusion Criteria

1. Patients with acute inferior wall infarction and unstable angina were excluded from this study.
2. Patients with symptoms of cardiac failure were excluded.

3. Patients with H/o. recurrent episode of tachyarrhythmia and brady arrhythmia were excluded.
4. Patients with ongoing chest pain, uncontrolled hypertension and any sign of active infection were excluded.
5. Patients older than sixty five years were excluded.
6. Patient with H/o upper GI bleed were excluded.
7. Patient who were not willing for Transesophageal study were excluded from the study.

Patient Characteristics

Among the total number of 106 patients, 86 patients were male and 20 patients were female. All the patients were presented with acute anterior wall myocardial infarction with an average median delay of 5 hours and 30 mts

All were thrombolysed with intravenous streptokinase 1.5 million units over a period of sixty minutes.

Surface ECG

Surface ECG was taken to all the patients. ECG was analyzed for the presence of anterior wall myocardial infarction. Subsequently serial ECGs were taken. The lead in which there was maximal ST elevation was noted. The percentage of this ST segment resolution after 90 minutes of thrombolysis was observed to document successful thrombolysis. Less than fifty percent resolution of maximal ST segment elevation at 90 minutes after thrombolysis was taken as unsuccessful or failed thrombolysis.

Trans thoracic Echocardiogram

In all the patients 2D, M mode, and Doppler examination were done by transthoracic echocardiogram. Left ventricular internal dimensions, wall thickness, ejection fraction and wall

motion abnormalities were recorded and analyzed in all patients.

Equipment

In our study we used ALOKA - SSD - 4000 system with multiplane Transesophageal echocardiographic probe. It generated two dimensional images at multiple frequencies (5 to 7.5 MHZ).

Instrumentation

In multiplane TEE the ultrasound beam was steered through 180 degree with physical manipulation by turning the appropriate knobs at the proximal end of the probe. An icon on the screen of the ultrasound machine in the form of semi circle. An needle like indicator in that semi circle array was steered. By convention 0 degrees will be shown on the left and 180 degree on the right of the semi circle. The needle moved in a counter clockwise direction when the beam was guided from 0 to 180 degrees.

The multiplane probe was interfaced to a standard ultrasound imaging console. Piloting to ultrasound beam in this fashion provides continuous visualization of the transition between adjacent structures which improves the operator understanding of the spatial relationship of the cardiac structure.

Procedure

- The patient was instructed not to eat in the proceeding 8 hours other than taking important medication with a sip of water.
- Informed consent was obtained in all the patients.
- Topical oropharygeal anesthesia with 2% lignocaine viscous was given to all the patients. Patients were instructed to gargle and keep it in the throat for 10-to 20 minutes.
- If the patient was very anxious, conscious sedation achieved with 1 to 2 mg of Inj.

Midazolam.

- The patient was then positioned in the left lateral decubitous position with head flexed forward into the chest. This position of the neck was important because it eased out the passage of the probe into esophagus. The hip and knees were flexed.
- Dentures if any were removed and the oropharynx was inspected in detail for loose teeth or other abnormalities.
- Intravenous access was obtained in all the patients.
- Oxygen saturation, blood pressure and cardiac rhythm were monitored and oxygen was given via nasal prong in select patients.
- Prior to starting the procedure the probe was inspected for any damage to the outer layer to prevent electrical and thermal injuries.
- The distal and of the probe was lubricated with lignocaine jelly and the bite block was inserted between teeth. The tip of the probe was partially flexed.
- The left index finger guide the probe avoiding entry into the lateral recesses with the right hand the probe was inserted into the mouth over the surface of the tongue in the midline toward the back of the throat.
- The patient was asked to swallow with gentle downward pressure applied at the same time using the finger to guide the scope keeps probe in midline and direct it toward the posterior pharyngeal wall and the upper esophageal sphincter.
- Using the control dial the scope was forced anteriorly, to negotiate the turn around the back of the tongue. Once the scope has passed from the oropharynx to the upper esophagus sphincter the probe then turned to the neutral or slightly retroflexed position to avoid the trachea inlet.

- The patient was asked to swallow and as the patient was swallowing scope was gently advanced through the upper esophagus sphincter. Swallowing enabled the sphincter to relax and gentle forward pressure on the shaft of the probe. Simultaneously resulted in the successful esophageal intubation.
- The probe was then advanced to about 30 to 40 cm from the teeth (incisors). Only minimal resistance should encounter in at the upper esophageal sphincter prior to its relaxation and passage of scope in the esophagus. Any unusual resistance or bowing of the scope suggested obstruction and the scope should be withdrawn and intubation reattempted. At no time the probe should be forced.
- The standard images were obtained from within stomach followed by as the probe was withdrawn into esophagus.
- An orderly sequence was followed for a complete study. First from the distal gastric fundus and then by stepwise withdrawal and imaging the probe was brought back to upper esophagus. At each transducer location the imaging beam was stared from O to 180 degrees in 15 degrees increment. Complete two dimensional sweep was followed by relevant color flow Doppler imaging.
- In our study a short axis view at the aortic level was obtained by rotating the image plane between 30° and 45° and withdrew the probe from lower esophagus to mid esophagus at aortic level.
- The origin of left main coronary artery was easily identified after minor adjustments in the depth and tilt of image plane. The right coronary artery was more difficult to visualize.
- At this position the following observations were made

  1. Visualization of left Main, proximal left anterior descending artery and, proximal left

circumflex artery and proximal right coronary artery.

2. Size, the peak and mean blood flow velocities in both diastole and systole in left main and proximal left anterior descending artery.

From the transesophageal or transgastric position the transducer was turned in either direction until the image plane was directed slightly left of the patient spine to obtain short axis view of the descending thoracic aorta. The aorta appeared circle and showed normal systolic pulsations.

TEE examination of the descending thoracic aorta was performed in all the 106 patients to find out the presence of atherosclerotic plaque. The thoracic aorta was considered normal when the intimal surface was smooth and continuous without luminal irregularities or echo destiny. If the intimal surface was increased in echo density but remained smooth and continue with luminal irregularities it was defined as grade 1. Intimal thickness less than 5mm with higher echogenic area disrupting the normal smooth surface of the vessel wall and cause lumen irregularities was classified as Grade II. Grade III changes consist of intimal thickness more than 5mm and or obvious lumen irregularities associated with localized highly echogenic mobile lesion protruding into the vessel lumen Grade II and Grade III lesions were considered to be atherosclerotic aortic plaque. Grade I lesions on would have a potential to develop atherosclerotic plaque.

The following observations were made at this position.

1. The size the descending aorta
2. Intimal thickness
3. Presence of atherosclerotic plaque and its size.

RESULTS

This study demonstrated high quality imaging of left main coronary artery and measurement of luminal diameter by using transesophageal echocardiography. Statistical analysis were done by using student T test and Chi Square test.

Among total number of 106 patients with acute myocardial infarction who underwent thrombolysis with Inj.Streptokinase satisfactory examination of full length of the left main coronary were obtained in all (100%) the patients. In addition to left main coronary artery the origin and proximal portion of LAD was seen in 98 of 106 patients (93%) and proximal portion of LCX was seen in 92 of 106 patients (87%). Proximal RCA was visualized in 31 of 106 patients (29%).

The mean diameter of the left main coronary was 0.39 ± 0.06cm; Proximal LAD mean diameter was 0.30 ± 0.04 cm; Proximal left circumflex mean diameter was 0.27± 0.11cm.

The mean length of the left main coronary was 0.99 ±0.30cm; proximal LAD was 2.2±0.77cm and proximal left circumflex was 2.7±1.1cm. 28 patients of 106 showed significant narrowing of left coronary artery.

Among these 28 patients 6 patients showed narrowing of left main coronary artery, the rest 22 patients showed narrowing of proximal of LAD. The mean diameter and length in those patients with left main narrowing were 0.24±0.14 cm and 0.86± 0.31cm respectively.

Coronary flow velocity could be measured in 98 of 106 patients (92%). It was not possible to get Doppler flow velocity signal accurately in 8 patients.

The following velocity profile was taken as normal value (in cm / sec).

		LM	LAD	LCX	RCA

Diastole	Peak	-	49 ± 20	40 ± 15	37 ± 12
	Mean	71 ± 19	31 ± 15	25 ± 8	26 ± 7
Systole	Mean	36 ± 11	23 ± 11	21 ± 6	21 ± 9

28 patients in whom significant coronary artery narrowing was visualized by TEE showed turbulence across narrowed site. Both peak and mean diastolic flow velocities were increased in these patients. Mean and peak coronary flow velocity in patients with LM narrowing were 1.22±0.17 m/sec and 1.52 ± 0.2 m/sec respectively against 0.42 ± 0.35 m/sec and 0.55 ±0.4 m/sec of patients without LM disease and 0.92±0.38 m/sec and 1.12± 0.42 m/sec in patients with LAD narrowing against 0.34± 0.29 an m/sec and 0.46± 0.35 m/sec in patients without LAD narrowing. Average peak and mean velocities of combined LM and LAD narrowing were1.2± 0.38 m/sec 0.99± 0.37 m/sec respectively as compared to 0.37 ±0.16 m/sec and 0.27 ±0.13 m/sec in patients without narrowing.

Risk factor analysis showed 21 of 106 patients were hypertensives and 37 of 106 were diabetic. Hypercholesterolemia was present in 8 patients. Family history of coronary artery disease was obtained in 15 patients. 55 of 106 patients were smokers. 21 patients were associated with two on more risk factors.

21 out of 28 patients with significant left coronary artery narrowing were having two or more risk factors (4 of 6 with left main narrowing and 15 of 21 with narrowing of proximal LAD).

ECG analysis showed presence of RBBB in 19 patients. 10 of 28 patients who showed increased turbulence in the left main and proximal LAD had RBBB. Rest of 78 patients 9 patients showed RBBB at the time of presentation. 1 of 4 patients who had LBBB showed left main narrowing. 3, 2 and 4 of 106 patients developed first, second and third degree heart block

respectively but none of them showed proximal left coronary narrowing or turbulence. Atrial fibrillation was present in 2 patients. 29 patients had non sustained VT . 13 of these 29 patients with NSVT showed coronary artery narrowing. 20 were in the successfully thrombolysed group, 9 patients were in the unsuccessful thrombolysis group.

Transthoracic 2D echo showed ejection fraction ranging from 0.19 to 0.55. The mean ejection fraction was 0.32.. Mitral regurgitation was present in 38 of 106 patients. Left ventricular thrombus was present in 11 patients. Pericardial effusion was present in 5 patients.

28 patients with significant narrowing of left main and LAD 10 patients had mitral regurgitation. Mild pericardial effusion was present in 5 patients. Thrombus was seen in 4 patients.

106 patients who were thrombolysed with Inj. Streptokinase were divided into two groups. First group had 67 patients who were successfully thrombolysed and 39 patients in the second group who had unsuccessful thrombolysis. The average coronary blood flow velocity in the first group was 0.78 ±0.42 m/sec (peak) 0.62± 0.37m/sec (mean) and it was 0.34 ± 0.27m/sec(peak) and 0.24 ±0.11m/sec (mean) in the second group.

Out of 106 patients in our study we could do CAG for 48 patients. 5 patients showed normal coronaries and 43 patients had CAD (Single Vessel Disease -25 ; Double Vessel Disease -8 ; Triple Vessel Disease -7 ; LMD -3) Among 6 patients with LM narrowing by TEE, 3 patients underwent CAG and all the three had LM stenosis . 19 patients with LAD narrowing by TEE underwent CAG out of which 15 had proximal LAD narrowing angiographically.

80 patients of 106 in our study showed atherosclerotic plaque. The average length of the plaque was 2.9 cm ± 0.06 cm and the average width was 0.55 ± 0.06 cm. The average intimal thickness was 0.3 ± 0.06 mm.

26 and 13 of 80 patients with thoracic aortic plaque were diabetics and hypertensives respectively. Family history of coronary artery disease was present in 11 of 80 patients, 7 of 80 patients had hypercholesterolemia. 48 of 80 were smokers. Two or more risk factors were present in 17 patients.

Among 43 patients with angiographically proven CAD, 34 patients had thoracic aortic plaque by TEE and 9 patients did not show any plaque. Out of 5 patients with normal coronaries by CAG 3 patients had aortic plaque and 2 patients showed no aortic plaque by TEE.

DISCUSSION

We subjected 106 patients with anterior wall myocardial infarction who were admitted in ICCU at GRH Madurai from January 2006 to January 2008. The principle aim of out study was to find out the ability of transesophageal echocardiography to visualize left main, proximal left anterior descending and proximal left circumflex coronary artery and to measure the size of left main coronary artery. We also aimed at measuring the blood flow velocities in these arteries.

The descending thoracic aorta was visualized in all the patients to find out the presence or absence of atherosclerotic plaque and if it was present its size and intimal thickness. We also looked for any correlation between atherosclerotic plaque in descending thoracic aorta as a marker of coronary artery disease.

The evaluation of coronary artery is challenging because of the size and location of these vessels. The left main and right coronary arteries and the proximal segments of left anterior descending and left circumflex arteries can be visualized during transesophageal echocardiography in most patients.

Youn HJ & Foster E et al in their study found out that using high frequency multiplanar and digital acquisition and display with or without contrast agent would be a promising method for evaluating coronary artery disease by visualizing the proximal coronary artery stenosis and coronary artery anomalies. They claimed TEE would be a valuable clinical tool in measuring coronary blood flow and contributed to understand coronary artery physiology.

M.A. Taams et. al. in a prospective study with 22 patients who had angiographically

proven left main coronary disease found both left main and left circumflex coronary arteries were visualized in all the patients (100%) but left anterior descending artery was visualized in only three patients (13%). Extent of left main stenosis was correctly diagnosed in eighteen patients (78%).

In our study the visualization of left main and left anterior descending were 100% and 94% respectively. The visualization of LAD was 94% as compared **Taams et al** study population were it was only 13%.

K. Yoshida et al in their study obtained adequate images of full length of the left main coronary artery and its bifurcation in 90% (60 out of 67) of patients by using TEE. TEE could also able to find out significant coronary artery narrowing of the coronary lumen in 91% (10 out of 11). Angiographically proven left main stenosis was seen in 11 out of 60 patients (18.3%) Blood flow in left main coronary to left anterior descending artery was demonstrated in 85% (57 out of 67) by color Doppler.

In our study the significant left coronary narrowing was visualized in 28 patient out of 106 (26%) almost similar to above study. According to **Kasprzk J.D et al,** TEE could visualize and measure blood flow in coronary ostia and proximal LAD in all (100 %) the patients which consist with our study.

160 consecutive patients were studied to assess the feasibility, sensitivity and specificity to visualize proximal coronaries by **Memmola et al**. The entire proximal left coronary artery was adequately imaged in 111 patients (70%). According to him stenosis was considered to be present if the plaque narrowing of the coronary lumen were observed. He identified the presence of stenosis in 6 out of 6 (100%) 50 of 53 (79%) of angiographically proven left main and left anterior descending artery respectively. Then he found the sensitivity and specificity of

TEE in identifying stenosis of LMCA and Proximal LAD were 100% and 98% and 79% and 89% respectively. He concluded TEE identification of proximal LCA was feasible in most patients and accuracy in identifying significant proximal stenosis was higher for left main coronary artery.

J.C. Tardiff et al. performed intraoperative multiplane TEE for 45 consecutive adults. The left main and its bifurcation was visualised in all the patients sensitivity and specificity for detection and coronary artery narrowing were 100% when results were compared with angiographic data. Visualisation of proximal, mid and distal LAD segments were possible in 69%, 31% and 16% of patients respectively. The sensitivity and specificity in visualizing, the significant narrowing in proximal LAD was 89% and 100%, and colour Doppler examination was found to be less useful in detecting stenosis in proximal LAD (52%).

Alam et al. in his study demonstrated left main coronary trunk in 30 of 32 patients by TEE. Of these 30 patients 10 had angiographically normal and 20 had stenosis of left main coronary arteries. The TEE study revealed no stenosis in 9 of 10 patients without LM narrowing and atherosclerotic lesion in all the patients with stenotic left main trunk (100%).

In our study 3 out of 6 patients with turbulence and increased flow velocity across LM underwent CAG and all of them (100%) had significant LM disease which correlated exactly with the study findings of **Memmola et al**. **J.C. Tardiff et al.** and **Alam et al .** It was nearly 91% and 96% from the studies by **K. Yoshida et al** and **Samdharshi TE et al** respectively.

Out of 22 patients with proximal LAD narrowing by TEE we did CAG for 19 patients and 15 (78.9 %) had proximal LAD lesion which was similar to the findings by **Samdharshi TE et al** where 11 (78%) of 14 patients showed stenoses involving the left anterior descending artery angiographically,

According to **Waller** the length of Left main coronary ranged from 1 to 25mm before bifurcation into LAD and LCX branches. The total length of LAD coronary artery measured from 10 to 13cm whereas left circumflex measured 6 to 8 cm.

Samdharshi TE et al. evaluated proximal coronary artery in 111 consecutive patients intraoperatively and coronary angiogram was done with in a week. Transesophageal echo visualized the entire length of left main artery and it was 0.2 to 2.2 cm (Mean 0.93 cm). He could able to visualize the length 0.2 to 2.2 cm of proximal LAD and 0.1 to 3.4 cm of proximal LCX in 103 patients (92%) and 0.1 to 4.5 cm of the proximal right coronary artery in 55 patients (49%) from bifurcation.

Cheemalapathi Saikrishna et al. in his study attempted to establish and normal dimensions of coronary artery segments during life in Indians and compared this with western population. In this study the mean diameter (in millimeters) of LMCA was 3.72 ± 0.65 in male and 2.72 ± 0.48 in female; Proximal LCX 2.82 ± 0.63 in males and 2.68 ± 0.59 in females. Proximal RCA 2.75 ± 0.6 in males and 2.55 ± 0.57 in females.

Kaimkhani et al. studied coronary artery dimensions in Pakistani population and compared this with caucasians mentioned in the literature. The mean diameter (in millimeter) of LMCA was 4.28 ± 0.82; proximal LAD mean diameter was 3.22 ± 0.74 and the proximal LCX 3.02 ± 0.75; the mean diameter and RCA was found to be 3.08 ± 0.78.

In our study the mean length a left main was 0.99± 0.30 cm; the average length of visualized proximal LAD was 2.2 ±0.77 cm and proximal LCX was 2.78 ± 1.1cm correlates almost same with the finding of **Samdharshi et al**. The mean diameter (in cm) of Left main in this study was 0.39 ± 0.06; proximal LAD 0.30 ± 0.04 and Proximal LCX was 0.27 ± 0.11 similar to the findings of the study by **Saikrishna et al.**

R.Erbel et al. analysed coronary blood flow by TEE in his land mark paper and found that the velocity measurement was dependent on the position of the sample volume in relation to the coronary luminal narrowing. The velocity was normal proximal to the stenosis, increased with in the stenosis and reduced distally. Thus the anatomy of the artery had to be known to accurately measure coronary blood flow velocity. This was done effectively by TEE.

Fumihiko Kajiya et al. in his study evaluated post stenotic blood flow velocity in nine patients with 75% to 90% stenosis of LAD during CABG surgery. LAD blood flow velocities measured several location distal to the stenosis. The post stenotic blood flow velocities were reduced in their diastolic components with rich systolic component. The velocity configuration in the post stenotic portion was characterized by the presence of reverse flow velocities and irregularity of the velocity pattern near the wall.

In contrast, **Takeshi et al**. studied coronary blood flow velocities in the distal LAD by transthoracic echocardiography. He compared patients with significant LAD stenosis and patients without stenosis and found that there was no significant change in peak and mean diastolic blood flow velocity in these two groups.

Thomas Theiunissen et al. made assessment of coronary artery stenosis in patients at risk by TEE. In this study the normal blood flow velocities (in cm/sec) were 38 ± 11 and 79 ± 19 in LMCA during systole and diastole respectively and 36 ± 11 and 67 ± 19 in LAD. Turbulence was the indicative of significant upstream stenosis. In this study the maximal to prestenotic velocity ratio was taken as Doppler criteria for significant stenosis. Pulsed doppler interogation of upstream of the zone of turbulence (Prestenotic area) demonstrated peak diastolic flow velocity of 0.47 m/sec. Velocities at the site of aliasing or turbulence was significantly increased with peak diastolic velocity of 1.4 m/sec. The maximal to prestenotic

blood flow velocity ratio was >2.9 indicative of stenosis.

In our study there was significant turbulence with coronary flow velocity more than 1 m/ sec in 17 patients 6 of 17 in the left main and 11 of 17 in the proximal LAD.

This is in contrast to **Hutchison Stuart J et al.** who found the basal mean flow velocities were greater only in left main ostial stenosis than in proximal or mid LAD stenosis and the basal flow velocities in the proximal LAD stenosis was lesser than that of mid LAD stenosis.

Non invasive echocardiographic measurement of coronary flow velocity also enable to differentiate TIMI 3 flow from TIMI-2 coronary reperfusion in patients with acute myocardial infarction before coronary intervention.

Souki Lee et al. evaluate coronary flow velocity in 46 consecutive patients with acute anterior wall myocardial infarction. Echocardiography and subsequent coronary angiography done in all the patients. LAD flow was present in both TIMI 3 and TIMI-2 groups. The diastolic flow velocity, peak distal flow velocities were significantly higher in patients with TIMI 3 flow and concluded better antegrade flow by colour Doppler. Less reduced antegrade flow velocity by pulsed Doppler in patients with TIMI 3 flow in acute anterior myocardial infarction which enabled non invasive differentiation of TIMI 3 flow from TIMI 2 flow in patients with acute myocardial infarction.

Iwakura Katsuomi et al. measured coronary flow velocity after myocardial contrast echocardiography. He found peak flow velocities and duration during systole were significantly lesser in patients with no reflow than those with reflow. Early systolic retrograde flow was frequently observed in patients with no flow but peak diastolic flow velocities were similar between these two subsets.

In our study both systolic and diastolic blood flow velocities were significantly reduced in 30 of 79 patients with acute anterior wall myocardial infarction. The possibility of TIMI 2 or slow flow could be assumed in these patients.

The detection of atherosclerotic aortic plaque by TEE and its significance and limitation as a marker of coronary artery disease was assessed by **Yoshihisa Matsuma et al**. The criteria used to diagnose atherosclerotic plaque on TEE were the presence of focally and linearly increased echo density of the aortic intima with presence of plaque. He studied 84 patients who had undergone coronary angiogram previously. The plaques were detected in 93% of patients with coronary artery disease and 53% of patients without CAD.

Magdy F Ismaeil et al. defined plaque as simple lesion where the intimal thickness less than 5mm and complex lesion where the thickness more than 5mm with raised protruding echo dense plaque within the lumen. 49 of 60 patients (82%) included in his study population had significant CAD. Among patients with CAD, 82% of patients with single vessel disease, 89% of patients with double vessel disease and 100% of patients with three vessel disease had significant plaque in their aorta.

Tribouilloy C et al. retrospectively analysed 105 consecutive patients with valvular heart disease to assess the value of TEE detection of thoracic aortic plaque for predicting coronary artery disease. In 19 patients with significant coronary artery stenosis, 18 had thoracic aortic plaque by TEE study. In contrast aortic plaque existed in only 10 of remaining 86 patients with normal coronary arteries or mildly atherosclerotic coronary lesions. There was a close relation between the degree of intimal changes and severity of CAD. In this study, the multivariable analysis of patient's age, sex, risk factor of cardiovascular disease, angina and TEE findings revealed that atherosclerotic plaque was the most significant independent

predictor of CAD.

According to **Khoury Z et al** the frequency, distribution, and severity of thoracic aortic plaques were evaluated by transesophageal echocardiography in 152 consecutive patients undergoing coronary arteriography. Coronary artery disease was defined as ≥ 50% stenosis of ≥ 1 major branch. Atherosclerotic plaques were detected in the aorta in 90 of the 97 patients (93%) with CAD, but in only 12 of the 55 patients (22%) with normal coronary arteries. Atherosclerotic plaques in patients with CAD were found predominantly in the descending aorta (in 93%) and been in the aortic arch (in 80%), whereas the ascending aorta was the least involved (in 37%). In the descending aorta, 58% of the plaques were complex (>3 mm thick, ulcerated, mobile, or calcified), and in the aortic arch, 40% of the plaques were so calcified. Complex plaques were not found in the ascending aorta.

GP Fazio et al, in his 41 of the 61 patients, obstructive CAD was detected by angiography in at least one vessel (> 50% left main coronary artery stenosis or > 70% stenosis in the left anterior descending, right coronary or left circumflex artery distribution). In 37 of the 41, atherosclerotic plaque was detected in the thoracic aorta by TEE. 20 of 61 patients had normal coronary angiographic findings or non obstructive lumen irregularities. In 2 of these 20 patients, plaque was detected in the thoracic aorta by TEE. The presence of aortic plaque by TEE study had a sensitivity of 90% and a specificity of 90% for angiographically proved obstructive coronary artery disease. The positive predictive value of aortic plaque for obstructive CAD was 95% and the negative predictive value was 82%. Hence he concluded that the detection of atherosclerotic plaque in the thoracic aorta by TEE appears to be a marker for the identification of obstructive coronary artery disease and deserves further investigation.

Yoshihisa Matsuma et al, Magdy F Ismaeil et al, **Khoury Z et al , GP Fazio et al** and

others found a strong association between aortic plaque and CAD, but in our study though we could find increased percentage of patients showed plaque in descending thoracic aorta there was no statistically significant correlation between the presence of aortic plaque and angiographically proved CAD. This was similar to the conclusion by **Irene Meissner et al,** and **Bijoy K. Khandheria et al,** who could not find aortic plaque as an independent risk factor for CAD.

CONCLUSION

1. Transesophageal echocardiography is a promising and effective non invasive diagnostic method of visualizing and measuring the size of proximal left coronary artery in patients with coronary artery disease.
2. TEE could visualize the LM in all the patients (100%). By TEE we can visualize proximal LAD and proximal LCX in 93% and 87% respectively.
3. Patients with acute AWMI who had unsuccessful thrombolysis showed significantly low coronary blood flow velocity by TEE compared to successfully thrombolysed group who had relatively higher coronary flow.
4. Patients with LM and proximal LAD narrowing diagnosed by TEE showed angiographic correlation with coronary narrowing in CAG.
5. Descending thoracic aortic plaque was present in 76% of patients with acute AWMI. There was no statistically significant correlation between the presence of plaque and the degree of presence and severity of CAD.

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ABBREVIATIONS

ACS	Acute Coronary Syndrome
AMI	Acute Myocardial Infarction
AWMI	Anterior Wall Myocardial Infarction
CAD	Coronary Artery Disease
CBFV	Coronary Blood Flow Velocity
CFR	Coronary Flow Reserve
CFV	Coronary Flow Velocity
FFR	Fractional Flow Reserve
LAD	Left Anterior Descending Artery
LCA	Left Coronary Artery
LCX	Left Circumflex Artery
LM	Left Main
RCA	Right Coronary Artery
SAX	Short Axis
TEE	Transesophageal echocardiography

PROFORMA

TEE - EVALUATION OF CORONARY ARTERY DISEASE

NAME : AGE : SEX IP.NO :

CD. NO: ADD : D.O.A :

CHEST PAIN
BREATHLESSNESS
PALPITATION

MEDIAN DELAY

RISK FACTORS : DM HT HYPERPIDEMIA SMOKING
PREVIOUS H/O OF MI FAMILY H/O CAD

PULSE : BP : RR :

JVP :

CVS -S1 S2 S3 S4 RS-CREPTS
MURMUR
RUB

CNS :FOCAL DEFICIT

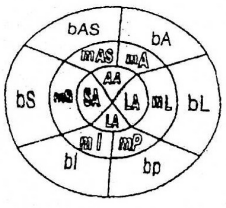
HB : TC : DC : ESR

ECG :
TYPE OF MI
ST SEGMENT ELEVATION SCORE
BUNDLE BRANCH BLOCK
AVR. ST SEGMENT
VI-ST SEGMENT
ARRHYTHMIAS

ECHO : M MODE

1. a) LVID (d) b) LVID (s) c) EF

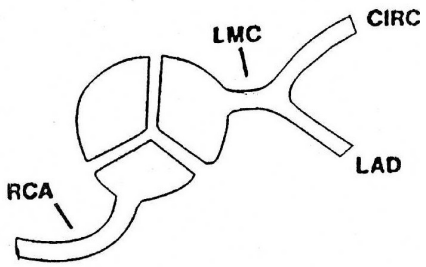
2. RWMA



3. MR
4. PERICARDIAL EFFUSION
5. LV CLOT
6. DIASTOLIC DYSFUNCTION

TEE :

CORONARY ARTERY EVALUATION



LEFT MAIN

- Size
- Flow
- Narrowing
- Doppler

PROXIMAL LAD

- Size
- Flow
- Narrowing
- Doppler

PROXIMAL LCX

- Size
- Flow
- Narrowing
- Doppler

PROXIMAL RIGHT CORONARY

- Size
- Flow
- Doppler

DESCENDING AORTA

- Size
- Intimal Thickness
- Plaque

ANGIOGRAM :

1. LEFT MAIN
2. LAD
   - TYPE
   - SEPTAL
   - DIAGONAL
3. CIRCUMFLEX
   - DOMINANCE
   - OM
4. RCA
   - DOMINANCE
   - PDA
   - PLB

LV ANGIOGRAM